Intravenous atropine treatment in infantile hypertrophic pyloric stenosis

Hypertrophic pyloric stenosis of infancy is a disorder of early infancy with typical clinical features and well-established radiological appearance of the pyloric canal. Many studies with surgical and medical treatment have been reported over the past fifty years. Pyloromyotomy has tended to become the favoured method of treatment as with expert paediatric, surgical, anesthetic, and nursing services and specialisation in some centres has been established in some series of cases. However, the interest has been low, since the surgery is widely accepted in European countries and the United States; it is however now becoming popular in Japan.

We are truly honoured to receive the comments of Dr Corner, who is a pioneering neonatologist and reported medical treatment with methyl scopolamine nitrate for infantile hypertrophic pyloric stenosis (IHPS) in 1955. She pointed out that methyl scopolamine nitrate might be better than atropine sulfate in terms of effectiveness and side effects. One of the reasons why atropine was used in our study is that methyl scopolamine is not available in our country. Scopolamine butyrylcholine is an available quaternary ammonium derivative of scopolamine and lacks toxic side effects. However, this agent tastes bitter and is difficult to give orally to infants. Therefore, this agent is only given intravenously in infants with IHPS.

We do not know if it is worthwhile to attempt combination therapy with intravenous scopolamine butyrylcholine and oral atropine rather than the intravenous and oral atropine therapy. Secondly, we already knew that an intravenous atropine injection of 0.01 mg/kg was effective enough to abolish transiently the phasic and tonic pyloric contractions characteristics of IHPS. We used an intravenous atropine injection of 0.01 mg/kg in our study to confirm that pyloric contractions were the cause of disturbed transpyloric flow in this condition by seeing that their inhibition with the dose of atropine ameliorated symptoms.

We agree with Dr Corner’s last comment, but believe that intravenous atropine therapy is possible not only in high level paediatric centres, but also in general hospitals where infusion therapy with intravenous atropine injections can be done safely in small infants. Clinical trials are now ongoing to establish more efficient treatment strategy for IHPS with medical and surgical therapy in our country.

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References

Hypothermia in a child secondary to ibuprofen

A 7 year old girl was admitted with right lower lobe pneumonia. On admission her temperature was 37.9°C. After five hours she received ibuprofen (6 mg/kg). Subsequent to this single dose her temperature decreased to 33.5°C (core temperature 34.9°C) over four hours. On examination her pulse was 90/min, blood pressure 90/50 mm Hg, SaO₂ 96% in air, and respiratory rate 20/min. Respiratory examination was consistent with signs of right lower lobe consolidation. The rest of the examination, including the central nervous system, was unremarkable.

Results of investigations included: Hb 125 g/l; white blood cell count 10.7 × 10⁹/ℓ; platelet count 81 × 10⁹/ℓ; C reactive protein 180 mg/l; blood glucose 4.6 mmol/l; Electrolytes and all other biochemical investigations were normal. Thyroid and cortisol assays were normal. Results of all tests to determine possible bacterial or viral aetiology were all negative (blood and urine culture, viral serology, and respiratory mycoplasma). Magnetic resonance imaging (MRI) of the brain was normal.

The hypothermia was so marked that we had to use a hot air spacer blanket to raise her temperature. Despite all the efforts she remained persistently hypothermic for four days (see fig 1).
Vagal overactivity: a risk factor of sudden infant death syndrome?

Since early 1990, the incidence of sudden infant death syndrome (SIDS) has dropped sharply because of public health campaigns aimed at educating parents and promoting the use of safe sleep practices. The marked decline in SIDS incidence has been attributed to raising awareness about the dangers of the prone sleep position, particularly among parents of very young infants. However, the precise mechanisms underlying this decline are not fully understood.

The prone sleep position is considered to be a primary risk factor for SIDS due to its association with higher rates of hypoxemia, oxygen desaturation, and apnea. These respiratory events may cause a hypercapnic ventilatory response, which can further reduce oxygen saturation and increase the risk of sudden infant death. Indeed, infants who have experienced recurrent episodes of hypoxia are more likely to experience SIDS, underscoring the importance of respiratory stability during sleep.

A recent study estimated the SIR separately among siblings of children with Vagal overactivity (VO) and a family history of SIDS. Despite the marked decline in SIDS, it is still the leading cause of postneonatal mortality. Better knowledge of other risk factors may allow identification of populations at higher risk and for the design of interventions to decrease infant mortality from SIDS through the implementation of appropriate prevention measures. Our findings suggest that VO may be involved in SIDS and that children with VO or a family history of SIDS may be a potential high-risk population for intervention.
Glucose metabolism in sleep disordered breathing

An association between sleep disordered breathing (SDB) and impaired glucose tolerance has been reported in adults. Although SDB has been reported in diabetic children, no data are available on glucose metabolism in children with SDB. We used glycated haemoglobin (HbA1c) for the preliminary assessment of glucose metabolism in paediatric SDB patients.

HbA1c was measured in 12 children aged 26–116 months (mean 63) with suspected SDB owing to adenotonsillar hypertrophy. Informed consent was obtained from the guardians of each patient, and consent was obtained from the child if older than 5 years of age. Overnight polysomnographic studies were performed once for each patient by the standard method described elsewhere. The desaturation time (percentage of total sleep time with oxygen saturation <90%), minimum oxygen saturation level, and apnoea–hypopnoea index (AHI) were calculated. Complete blood count, blood gases, and blood chemistry (glucose, total protein, albumin, uric nitrogen, creatinine, uric acid, sodium, chloride, potassium, calcium, phosphate, lactate dehydrogenase, glutamic oxaloacetic transaminase, glutamic pyruvic transaminase, γ-glutamyl transpeptidase, alkaline phosphatase, total bilirubin, total cholesterol, and triglyceride) were also determined. The patients had no respiratory failure, heart failure, or coma. None of their weights exceeded 120% of their ideal weight for their heights. Desaturation time clearly divided the patients into two groups: six patients whose desaturation time was 0 or 0.1 (mild SDB group); and six whose desaturation time exceeded 4.0 (severe SDB group). The average HbA1c value for the severe SDB group (5.0, SE 0.07) was significantly higher than that for the mild SDB group (4.6, SE 0.10) (p = 0.01), although the actual HbA1c values were all within normal range. No other items showed significant differences between the two groups.

The severity of respiratory disturbances during sleep in diabetic children has been known to correlate with the duration of diabetes and with the HbA1c value. Recently, SDB parameters were found to be associated with worsening insulin resistance independent of obesity in adults. The current study shows that serum HbA1c is increased in association with the degree of desaturation in non-obese paediatric SDB patients; HbA1c levels should, however, be monitored after treatment. SDB and glucose metabolism are hypothesised to be closely associated in children as well as adults.

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5. Kohyama J, Hasegawa, J S Ohinata. Department of Pediatrics, Faculty of Medicine, Tokyo Medical and Dental University, Japan.

Short versus standard duration antibiotic treatment for UTIs: a comparison of two meta-analyses

Having recently published a meta-analysis on the same clinical question, it was with great interest that we read Michael et al’s systematic review of short versus standard duration antibiotic for urinary tract infections (UTIs) in children. Given the publication (in close succession) of two meta-analyses on the same question with (on the surface) strikingly different results, we thought a comment was in order.

First, we applaud the authors on their methodologically sound review. The literature search was explicitly described and exhaustive. In fact, the authors identified a few studies that we had missed. “The study outcomes for meta-analysis (frequency of positive urine cultures at 0–7 days after treatment and at 10 days to 15 months after treatment, and development of resistant organisms and recurrent UTI) were relevant and clearly defined.”

The authors provided appropriate and important meta-analysis measures including summary relative risks (RRs) and a quasi-NNT calculation with varying risk of treatment failure in the standard treatment group and confidence intervals corresponding to “best” and “worst” case scenarios.

For their primary outcome, frequency of positive urine cultures 0–7 days after treatment, the authors found no significant difference between short (3–5 days) and standard (7–14 days) treatment (RR 1.06; 95% CI 0.64 to 1.76). This is in contrast to our finding of a 94% increased pooled risk of treatment failure with short course treatment (≤3 days) compared to standard treatment (7–14 days) (RR 1.94, 95% CI 1.19 to 3.15; NNT=15, 95% CI 100 to 7). Why the discrepancy? We postulate a few possible explanations. Thus, any child who has unexplained encephalopathy, regardless of its cause and clinical setting, should be screened for MCADD.

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Our omission of certain studies identified by Michalea and colleagues may have biased our results. However, of the three studies that we missed and that they included in their analysis of treatment failure at 0–7 days after completion of treatment, two favoured standard duration treatment, which would have supported our pooled RR result. Another possible explanation for the discrepancy was the use of different definitions of treatment failure. For our definition of treatment failure we pooled persistent infection with failure to eradicate the organism within 2 days of initiation of treatment and relapse (recurrence of symptoms and reinfection within 2 weeks of cessation of treatment after initial bacteriologic cure), whereas Michael et al used frequency of positive cultures 0–7 days after cessation of treatment as their primary outcome measure of treatment failure. If reinfections later than 7 days after cessation of treatment occurred more often in recipients of short-course treatment, then Michael et al’s definition of treatment failure could have failed to capture the therapeutically relevant advantage of standard duration treatment.

However, the most likely explanation for the divergent results was the different ways in which the study question was framed and the resulting differences in studies included in the meta-analyses. We compared ≤3 days of treatment to 7–14 days of treatment, whereas Michael et al compared 2–4 days of treatment to 7–14 days of treatment and excluded 11 studies comparing single-dose or single-day treatment to standard duration treatment.

The reasons for this exclusion are unclear, although we presume that they felt single-dose or single-day treatment was not a fair comparison with 7–14 day treatment. However, a number of randomised controlled trials (RCTs) made this comparison, suggesting that clinicians are, in fact, interested in the potential efficacy (and significantly increased case of adverse events) of single-dose or single-day treatment. Inclusion of these studies in our analysis strongly influenced the pooled risk of treatment failure with short-course treatment. When we excluded these studies in a sub-group analysis of 3-day versus long course (7–14 day) treatment, the risk of treatment failure fell to 1.36 (95% CI 0.68 to 2.72) (NNT=50; 95% CI 33 - 13).

Thus, our meta-analysis demonstrates clearly that single dose or single day antibiotic treatment is not as effective as long-course treatment for UTIs in children. The two meta-analyses together suggest that: (1) “longer” short-course therapies may be as effective as 7–14 days of antibiotics and
(2) there is probably a duration of treatment threshold for “short-course” antibiotic treatment, above which longer duration of treatment confers no therapeutic advantage. Michael and colleagues suggest that as little as 2 days of treatment may be sufficient. However, only one of the trials in their meta-analysis studied 2-day treatment and that only ensured long-course treatment with a RR of UTI 0–7 days after completing short course treatment of 2.17 (95% CI 0.48 to 9.76). The duration of treatment threshold may be 3 days, but the point estimate of relative risk of treatment failure with 3 day treatment in this meta-analysis suggests otherwise. If the duration of short-course treatment for which there is no difference in efficacy compared with standard therapy is actually greater than 3 days, then the added convenience and cost savings of “short-course” treatment become marginal. In the absence of appropriately powered RCTs (or meta-analyses) examining outcomes (treatment failure, reinfection, emergence of resistant organisms and cost) with “longer” short course treatment regimens (3, 4, and 5 days), we think that clinicians should continue to treat UTIs in children with at least 7 days of antibiotics. 

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References


Table 1

<table>
<thead>
<tr>
<th>Systematic review</th>
<th>Comparison of duration of therapy</th>
<th>Number of data sets</th>
<th>Risk for persistent bacteriuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tran et al., 2001</td>
<td>1–4 days v 5–7 days</td>
<td>13</td>
<td>RD = 4.26 (95% CI 0.95, 9.48)</td>
</tr>
<tr>
<td>Keren &amp; Chan, 2002</td>
<td>3 days v 7–14 days</td>
<td>5</td>
<td>RR = 1.36 (95% CI 0.68, 2.72)</td>
</tr>
<tr>
<td>Michael et al., 2002</td>
<td>2–4 days v 7–14 days</td>
<td>8</td>
<td>RR = 1.06 (95% CI 0.64, 1.76)</td>
</tr>
</tbody>
</table>

*RD, risk difference; CI, confidence intervals; RR, relative risk

Because there is no significant difference between short duration and standard duration treatment in the number of children with persistent UTI after treatment, it is not possible to calculate a number needed to treat to prevent one episode of persistent bacteriuria. From our systematic review, we are not able to determine whether there is an “optimum duration of treatment threshold” as postulated by Keren and Chan. Only one study included in the meta-analysis, examining the effects of short duration and standard duration treatment in clearing bacteriuria, compared 2 days of treatment with 10 days’ treatment. In their letter above, Keren and Chan argue that this study favours short duration treatment. However, there was no significant difference between treatments in the number of children with persistent bacteriuria at the end of treatment (RR 2.17; 95% CI 0.48 to 9.76) although the wide confidence intervals do not exclude the possibility that short duration treatment could be more or less effective than standard duration treatment. No significant differences in the number of children with persistent UTI after treatment between short duration and standard duration antibiotic treatment have been found in three systematic reviews of randomised controlled trials despite different study inclusion criteria and definitions of persistent infection. As addressed in our review, the wide confidence intervals around the summary estimates indicate residual imprecision in the results. However, this statistical imprecision is of doubtful significance for most children, who are at a low risk (1–3%) of persistent UTI at the end of treatment following their first lower tract UTI. Therefore, we do not support Keren and Chan’s conclusion that clinicians should continue to use short tract UTI with standard duration treatment. Instead, we believe that short duration treatment may be used to treat children with lower tract UTI.

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References


www.archdischild.com
Is life long follow up for patients with Kawasaki disease indicated?

Brogan et al recommended life long follow up for patients with Kawasaki disease, including those who do not have coronary artery involvement. The reason they quoted was to document the blood pressure and provide general advice regarding other risk factors. The American Heart Association recommends echocardiographic (EKG) evaluation of the coronary arteries at presentation and follow up ECG at 6–8 weeks and 6–12 months after the onset of symptoms for those who did not have or just have transient coronary artery involvement. They do not recommend follow up after first year unless cardiac disease is suspected.

Tuohy et al demonstrated, in their multi-institutional review of 536 patients, that no patient with a normal follow up ECG, performed within 2 months following disease onset, subsequently developed echocardiographic coronary artery abnormalities. Even those patients with initial echocardiographic abnormalities that became normal at 1–2 months remained normal thereafter. Scott and colleagues showed that no patient with a normal EKG at 2 weeks to 2 months after the onset of symptoms had subsequent ECGs that revealed significant coronary artery abnormalities and questioned the value of 6–12 month ECG in the same group.

Brogan et al did not make any comments about the adverse effects of life long follow up, such as anxiety and inappropriate restriction of activities. Finally, there were no comments about the cost and resources for providing life long follow up. The authors did not specify whether paediatric cardiology, general paediatrics, or general practitioners would follow up; all of them already have increasing workload.

References

Management of childhood osteoporosis

I read with interest this recent review article that summarises current knowledge about this subject. I have a number of comments that are pertinent to the discussion. As the authors allude to, there is currently a lack of good evidence on which we can base preventative management, although calcium and vitamin D supplements are routinely used by some paediatric rheumatologists, there appears to be only one short term study suggesting this may be beneficial for bone density. The problem is in relation to growth hormone therapy are methodologically flawed because neither have accounted for the change in apparent bone density, which will occur in any child who grows better for any reason when assessed by modalities such as dual energy x ray absorptiometry.

As illustrated by another article in the August 2002 edition of Archives, there is a lack of good evidence on which to base much paediatric management and it is imperative that further research, especially randomised controlled trials, is undertaken in the area of prophylaxis against osteoporosis in children with chronic disease on steroids. Paediatric endocrinologists will be familiar with the flurry of small uncontrolled studies undertaken in numerous groups of children with short stature when recombinant growth hormone became available. Many reports of short term improvements in growth velocity have not been supported by long term outcomes in height. There is a risk that a similar phenomenon will occur with the use of bisphosphonates in children with chronic disease and low bone density without properly designed studies and satisfactory outcome measures.

The use of glucocorticoids in children with chronic disease can account for a decrease in bone density due to a decrease in bone mass. Paediatric endocrinologists and I would argue strongly that the management and prevention of osteoporosis requires specialist expertise just as the management of growth retardation currently does. It is important that in each tertiary centre such a specialist service is provided by one department that has expertise in the interpretation of bone density scans in children and the management of children with osteoporosis. Such individuals may not only be paediatric endocrinologists but may also be a paediatric rheumatologist, a general paediatrician with a special interest in bone disease or a metabolic bone disease specialist. It is only in this way that we can learn more about the management of this condition and avoid children being treated inappropriately.

References

Newborn screening for Duchenne muscular dystrophy

Elliman, Dezauteaux, and Bedford, in their recent leading article on newborn and childhood screening, include reference to newborn screening for Duchenne muscular dystrophy (DMD). They argue that the main value of such a screening programme is to warn parents that future sons may be affected, and support this statement with reference to Jarvinen et al. This paper does not report a newborn screening study but the results of a retrospective study of 23 females in Finland carrier tested for DMD during childhood. However, a newborn screening programme for DMD has been running in Wales since 1990 (900–8 as a research evaluation and from 1998 health authority funded). During the research period interim evidence was published. More recently the full results of our prospective study have been published. Our evaluation has demonstrated that a newborn screening programme for DMD can be acceptable to both parents and health professionals, providing that a rigorous service delivery protocol is in place and the programme is supported by an effective infrastructure, in particular by paediatric and genetic services.

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References

www.archdischild.com
The effect of sanctions on children of Iraq

Sanctions were imposed on the people of Iraq in 1990. Iraqi people are still suffering, especially children. Infant mortality (IM) has increased more than five times. Previously it had decreased from 139 in 1960 to 20 in 1989, which was comparable to developed countries. In 1992 it went up to 111.1 In 1999, a decade later, IM was still high at 104.2 The Gulf War and trade sanctions caused a threefold increase in mortality among Iraqi children under 5 years of age. It has been estimated that more than 46,900 children died between January and August 1991.3

The study of the UN Food and Agricultural Organisation, published in a letter to the BMJ in 1995, concluded that deaths of more than 560,000 children could be attributed to UN sanctions. It also stated that the death rate among children under 5 years in Baghdad had increased fivefold since the war ended in 1991.4 Data for 1994–99 showed that mortality in children under 5 years was 131 per 1000 live births, compared with 56 for 1984–9before sanctions. The reasons for excess deaths are clear—economic collapse, lack of safe water, and inadequate sanitation, lack of safe water, and inadequate

The rate of low birth weight (<2500 grams) which was in the region of 9% in the period 1980–1987 increased to 21% in 1994.5 The 1995 Baghdad nutrition survey of children under five years of age showed that the percentage of children below <2SD in urban Baghdad was 28% for stunting, 29% for underweight, and 12% for wasting.6 Malnutrition (hence <2SD) was noted among children, 10% for stunting, 7% for underweight, and 3% for wasting.7 The survey by FAO in the year 2000 indicated the prevalence of wasting in children under 5 years of age at the unacceptable high level of 10%, only a marginal difference from the 1995 survey.1

In school children aged 6–8 years the prevalence of wasting ranged from 1% in the upper class to 6.7% in rural areas. Similar differences were found for stunting and underweight.8 In a 1994 survey 1.6% of children under 5 years were reported to have night blindness, indicating vitamin A deficiency. A survey of school children in the north in 1994 showed a 30–50% prevalence of goitre, and evidence of iodine deficiency disease elsewhere throughout the country.9

Diarrhoeal diseases and mortality due to dehydration were well under control prior to the Gulf War; there was a threefold increase from May 1990 to May 1991.10 Other water borne infections increased from 1990 to 1999, for example typhoid by 60% and cholera almost fivefold.11 A measles epidemic occurred in 1998.12 There were alarming rises in cases of malaria and leishmaniasis. Other infections like tetanus, poliomyelitis, diphtheria, and pertussis all showed an increase after the Gulf War.1,5

The National Immunization Programme which had begun in 1985 came to a complete halt between January and April 1991.13 The percentage of fully immunised one year old children fell from 94 for tuberculosis, 83 for diphtheria, tetanus, and pertussis, 83 for polio, and 82 for measles to 79, 63, 64, and 68 respectively.14

A child psychology study (1991) revealed a level of psychological stress and pathological behaviour that was the highest the authors had seen in 10 years of conflict research. It revealed a highly disturbed population of children. Fear and anxiety were associated with memories of crisis. Seventy five percent felt sad and unhappy, and four out of five expressed fear of losing their family by death or separation.1

There was a threefold increase in leukaemia in the southern provinces, sites of the Gulf War battlefield. A WHO investigation in 1995 suggested a possible link to products—now banned—of the familiar international food corporation which derived from depleted uranium used in piercing artillery shells. There were staggering deficiencies in cancer treatment facilities because of UN sanctions which were intended to exclude food and medicines.15 A report in 1996 showed that one third of hospital beds were closed. More than half of all diagnostic and therapeutic equipment was being sold. The medical community had experienced serious problems with lighting, cleaning, water supply, and sewage. The population had been burdened by a rapid rise in serious infections, nutritional deficiencies among children and pregnant women, and other treatable conditions for which neither drugs nor operations were available.16

Paediatricians have been isolated by the intellectual embargo from the international medical community. Physicians who wish to attend international conferences face travel restrictions, like denial of visas to European countries or the USA. In 1990, the delivery of European and American medical journals were abruptly stopped. This intellectual embargo served to undermine the care of patients, and denies Iraqi doctors the right to share scientific advancement and its benefits.17

Casano et al describe a case of refractory pulmonary hypertension with severe Bordetella pertussis infection. Their description of the literature is incomprehensible. In four cases of fatal pulmonary hypertension (PHT) in a series of 13 critically ill infants with B. pertussis,1 they noted that many of the patients had HIDS, as well as other diagnoses. The patients who developed PHT all were hypoxic, had failure to thrive, and were refractory to all currently available modalities including extra-corporeal membrane oxygenation. Hyperkalemia was an independent predictor of death when corrected for presentation delay, hypoxia, and previous use of insulin. The histological evidence was such that extreme leukocytosis predisposes to the formation of lymphocyte aggregates in the pulmonary vasculature that increased pulmonary vascular resistance via obstruction rather than

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hypoxic vasoconstriction. Therefore Dr Casa-
ño’s recommendation for the early use of
pulmonary vasodilators is unlikely to be suf-
ficient in this context. We are assessing the
impact of strategies aimed at reducing
lymphocyte numbers and adhesion in addi-
tion to standard treatments for pulmonary
hypertension.

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Authors’ reply
As Peters comments in his letter, we know that
hyperleukocytosis has been postulated as a
factor for pulmonary hypertension in Pertussis
infection, but necessary brevity did not make it
possible to report. Nevertheless, our patient
never reached these values of leucocytosis; it’s
possible, as in many other diseases, that
several pathogenic mechanisms contribute to
pulmonary hypertension, making a concomi-
tant treatment approach necessary.

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CORRECTIONS
In the paper by Clarkson and Choonara in the
December issue of ADC (Arch Dis Child 2002;87:462–7) the following corrections
have been noted:

Results; first sentence: there were 331
deaths with 390 suspected drugs (not 390 and
389 respectively as stated in the paper).

Results; section “Corticosteroids”: the third
sentence starting “No details were avail-
able...” should be deleted.

Results; section “Non-steroidal anti-
flammatory drugs (NSAIDs)”: the second
sentence “All reports for NSAIDs have oc-
curred since 1990” should be deleted.

Discussion; fifth paragraph: the penulti-
mate sentence should be “as recently as 1999
our study found a single fatality” (not 2
reported fatalities).

Discussion; fourth paragraph, second sen-
tence. The word “seven” before “cases” should
be deleted.

The journal apologises for the errors.

The following figure should have appeared
with the letter by Desai and Babu in the
October issue of ADC (Arch Dis Child 2002;
87:357).

Figure 1 Scimitar syndrome. Chest x ray
showing a curvilinear density which extends
from the right hilum towards the right
hemi-diaphragm which represents the
anomalous pulmonary vein.